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| **Unit 2: Cells & Cell Processes** | | | | | | |
| Name: | Start Date: | | | 11/29/16 |  |  |
|  | Quest Date: | | | 12/15/16 (Gamma) 12/16/16 (Epsilon) |  |  |
| Period: (Honors) | Teacher: Ms. J | | | |  |  |
|  |  |  |  |  |  |  |
| **Cells & Cell Processes** | Submitted | Resubmit | Correct | Evidence of Learning | Page | Date |
| **Objective 10:** Explain how genetic and environmental factors can influence the cell cycle and lead to cancer. |  |  |  | Catalyst: KWL Chart |  |  |
|  |  |  | Notes: Cancer |  |  |
|  |  |  | HW: p53 Article |  |  |
|  |  |  | Catalyst: TBD |  |  |
|  |  |  | HW: Video Questions “Cancer as a Genetic Disease” |  |  |
|  |  |  | HHMI Cancer Patient Data Activity |  |  |
|  |  |  | Types of Cancer Jigsaw Activity |  |  |

**Unit 2: Cells & Cell Processes**

Start Date: 11/28/16 Quest Date: 12/16/16

**Objective 9:**  Analyze how cells grow and reproduce in terms of interphase, mitosis, and cytokinesis.

*Essential Question:* What is the cell cycle?

*Essential Question:* What is the sequence and function of mitosis?

*“I Can” Statements:*

* Describe/outline the stages of the Cell Cycle: Growth 1, Synthesis, Growth 2, Mitosis, and Cytokinesis
* Organize diagrams of mitotic phases and describe what is occurring throughout the process

**Objective 10:** Explain how genetic and environmental factors can influence the cell cycle and lead to cancer.

*Essential Question:*  What is cancer?

*Essential Questions:* What causes cancer?

*Essential Questions:* How are different cancers caused, treated, and prevented?

*“I Can” Statements:*

* Describe the relationship between mutations in the DNA and uncontrolled cell growth
* Use an example of a specific cancer to illustrate how cancer is caused, treated and prevented

**Objective 11:** Explain how instructions in DNA lead to cell differentiation and result in cells specialized to perform specific functions in multicellular organisms.

*Essential Question:* How do cells become specialized?

*“I Can” Statements:*

* Explain that cells differentiate and give examples of differentiation/specialization
* Describe stem cells as undifferentiated and discuss how this is important to scientific research
* Explain the relationship between DNA expression and the type of cell that develops through differentiation

**Catalyst 1:** Fill out the following chart including what you already know about cancer, what you want to know, and by the end of the week we will fill out the L column concerning what you learned.

|  |  |  |
| --- | --- | --- |
| **Know** | **Want to know** | **Learned** |
|  |  |  |

**Catalyst 2: TBD**

Biology I (Honors) Name: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Notes: Why Cells Divide Period: \_\_\_\_ Date: \_\_\_/\_\_\_/\_\_\_

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Biology I (Honors) Name: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Activity: p53 and Cancer Period: \_\_\_\_ Date: \_\_\_/\_\_\_/\_\_\_

**Introduction**

## Tumor Suppressors: p53

The *p53* (or *TP53*) gene was discovered in 1979 and has emerged as one of the most important cancer-related genes to date. The gene, located on chromosome 17, produces a protein product that functions as a transcription factor . The genes controlled by p53 are involved in cell division and viability. Like the other tumor suppressors, the p53 protein functions to prevent unregulated cell growth.

The p53 protein interacts directly with DNA. It also interacts with other proteins that direct cellular actions. When DNA damage or other cellular insults are detected, p53 has the power to trigger cell death or apoptosis. The crucial role of p53 in maintaining proper control of cellular processes is underscored by the fact that the *p53* gene is found to be defective in about half of all tumors, regardless of their type or origin.[10](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote10_8006dxg)[36](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote36_krg36hj)The mutations that inactivate *p53* may be acquired during the lifespan of an individual (sporadic mutations) or they may be inherited. transcription.

## p53 Function

The p53 protein plays an integral role in the cell and is normally present in all cell types. The protein is localized in the nucleus where it functions as a transcription factor. The p53 protein is at the center of a large network of proteins that 'sense' the health of a cell and cellular DNA. The p53 protein is the conductor of a well orchestrated system of cellular damage detection and control. When damage is sensed, the activity of the p53 protein aids in the decision between repair and the induction of cell death (apoptosis).[41](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote41_l7b9un7)

As a transcription factor, p53 stimulates the transcription of a group of target genes. Among them, *p21* is one of the most important. The product of the the *p21* gene is a negative regulator of cyclin-dependent kinases, enzymes that are critical in the progression of the cell cyle and ultimately cell division.[10](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote10_8006dxg)By stimulating the transcription of the *p21* gene, p53 prevents cell proliferation. This stoppage gives the cell the opportunity to make repairs, if possible. If substantial DNA damage has occurred, the p53 protein can help to trigger cell death. The death of a cell that has incurred substantial DNA damage is beneficial to the organism because it prevents cells with deleterious mutations from proliferating.

As discussed in the introduction to this section, all cancer cells contain mutations in combinations of tumor suppressors and oncogenes. The removal of functional p53, the 'guardian of the genome,' from a cell allows for the accumulation of even more DNA damage and the division of cells that contain damaged DNA.

The mutation of the *p53* gene is one of the most frequent genetic changes seen in cancer cells. In addition to mutations that arise during the growth and development of individuals (sporadic mutations), there are forms of cancer associated with the inheritance of a damaged version of *p53*. One such syndrome, the Li-Fraumeni cancer family syndrome, is associated with a wide variety of cancers.[42](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote42_4koi3q0)In addition, several viruses have evolved ways of inactivating the p53 protein including the human papillomavirus, the causative agent of cervical cancer.

Due to the central role played by this protein in the regulation of cell division,  is a large amount of current research is committed to developing a safe method of restoring *p53* gene function.

In 2014, researchers discovered that an alternatively spliced form of p53 exists which works to DRIVE cancer development and spread.  The new form is called p53Ψ. The work was done in cell culture as well as with mice and rabbit antibodies.[43](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote43_qky9kw2)

**A Closer Look at Abnormal p53 and Cancer Development**  
A cell lacking functional p53 may or may not become cancerous, and correspondingly, a cell with normal p53 function may eventually lead to the formation of a cancerous growth. As discussed in the section on [Mutation](https://www.cancerquest.org/cancer-biology/mutation), to become cancerous, several different changes to the DNA of a cell must occur. One of the functions of p53 is to monitor the status of the cell's DNA. Along with a host of additional proteins, p53 helps to recognize and effect repairs to damaged DNA. The responses to damaged DNA include repair, cessation of cell division and cell death. Damage to the *p53* gene does increase the likelihood of cancer development. Remember that since p53 is a tumor suppressor, both copies of the gene must be inactivated in order to see the full effects. There are several ways in which p53 can be inactivated:

***Mutations***  
Alterations in the *p53* gene have several different effects on the activity of the gene, depending on the location of the alteration.

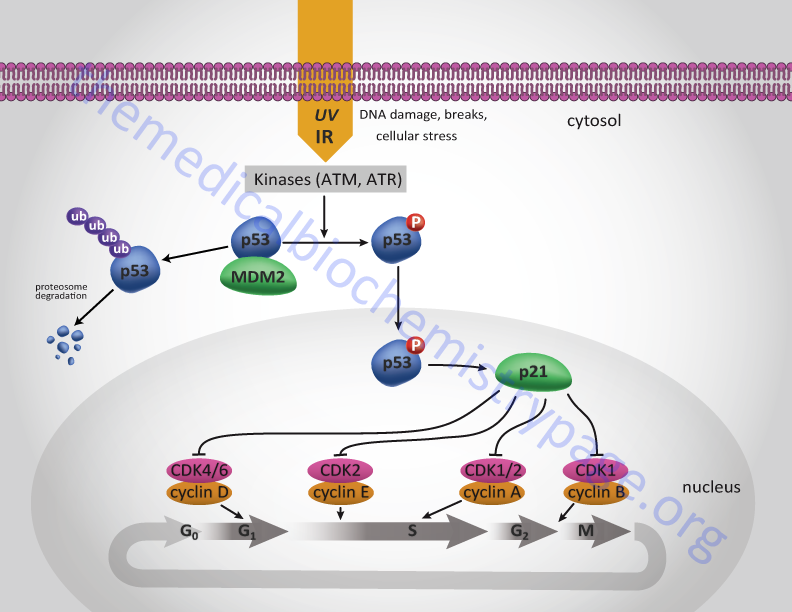
1. Mutations may occur in regulatory regions. These portions of the gene control how often, and when, the gene is transcribed (this region is called the promoter). A mutation in the promoter region can result in a decrease or absence of p53 in the cell.[44](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote44_l1jyxkw)
2. Mutations that occur in the protein coding region of the gene can impact the expression of the gene (or activity of the protein) in several ways:

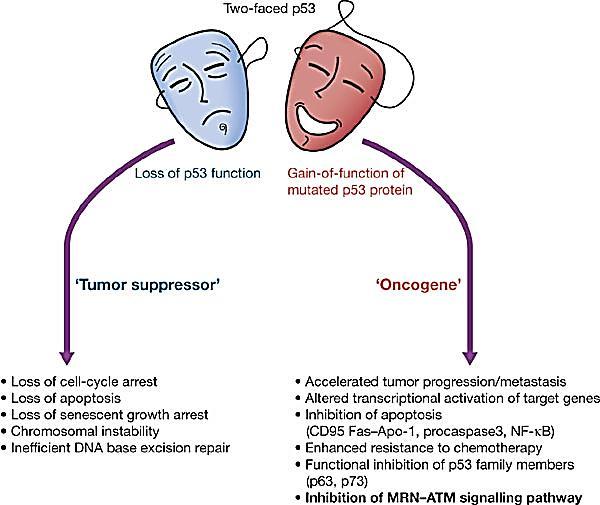
* A decrease in the activity of p53 as a transcription factor. The expression of the target genes of p53 that would be affected include *p21* (a protein involved cell cycle regulation), *Bax* (a protein involved in the induction of apoptosis), and thrombospondin-1(an angiogenesis inhibitor).[45](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote45_izs0wf3)[46](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote46_k3hddxl)
* A change in p53 that makes it more susceptible to degradation. If the p53 proteins in the cell are being degraded at a higher-than-normal rate they will not be able to perform their functions as tumor suppressors.[47](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote47_6dklo0j)

***Viral Inactivation***  
One of the functions of p53 is in 'guarding' the genome. Infection with viruses introduces foreign DNA into cells. p53, along with other proteins, is responsible for the cell's response to the presence of foreign DNA. Again, the responses include shutting down cell division and cell death. To avoid these responses, several different viruses have evolved ways of inactivating the p53 protein. An example of this is Simian Virus 40 (SV40) Upon infection with SV40, viral proteins are produced within the cell cytoplasm. One of the proteins produced is termed the Large T antigen. A function of this protein is the binding and inactivation the p53 protein. Other viruses such as Hepatitis and Human Papillomavirus produce similar proteins.

The elimination of functional p53 from the cell clears the way for cell division even in the presence of DNA damage. In the absence of p53, genetic instability as evidenced by increased mutations and aneuploidy are likely to increase. The increase in genetic damage leads to the accumulation of defective tumor suppressors and oncogenes.[48](https://www.cancerquest.org/cancer-biology/cancer-genes#footnote48_zwbblkz)

Weblink for review of the p53 gene: http://media.hhmi.org/biointeractive/click/p53/01.html





**Analysis Questions:**

1. Mutations in the p53 gene appear to be a major factor in the development of human cancers. Discuss the normal cellular functions of the p53 gene product and how alterations in these functions can lead to cancer.   
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
2. What is apoptosis? Why is the cell death associated with apoptosis desirable? How is apoptosis regulated?   
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
3. Add annotations to the diagrams on the back of the first page explaining the illustrations.

Biology I (Honors) Name: \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

Types of Cancer Jigsaw Read Period: \_\_\_\_ Date: \_\_\_/\_\_\_/\_\_\_

**Part I. Article**

*Read your assigned article and answer the questions below.*

Article Title:\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

1. What type of cancer is discussed in your article?  
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
2. What causes the development of this type of cancer?  
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
3. Are there steps that can be taken to prevent this type of cancer? Or are there genetics screenings to prevent it?  
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
4. What are the risk factors for this cancer? What type of people are at the highest risk?  
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_
5. What is the most common method of treatment for this cancer?  
   \_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_

**Part II. Jigsaw Reading.**

Fill out the chart to include information about the other articles your classmates read.

|  |  |  |  |
| --- | --- | --- | --- |
| **Type of Cancer** | **Causes** | **Risk Factors** | **Prevention?** |
|  |  |  |  |
|  |  |  |  |
|  |  |  |  |